1 Introduction

History of models and estimation attempts

Usefulness of bound: benchmark algorithms, experimental design, insight about inherent uncertainty – which do we address

2 Background

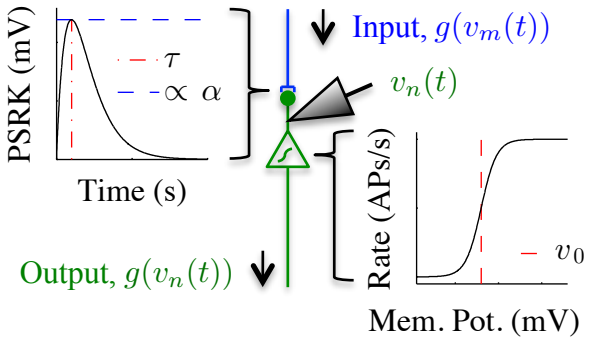

Figure 1: ***Neural Mass Model.*** *The somas of the neural mass are represented by the triangle. The synaptic connections are represented by the circle. The input mean firing rate, $g(v_m(t))$, is convolved with post-synaptic response kernel (PSRK), shown in the inset on the left, to give the mean membrane potential, $v_n(t)$. This is transformed by the sigmoid, shown in the inset on the right, to give the output mean firing rate, $g(v_n(t))$.*

Neural Mass Model Neural mass models map from a mean firing rate of a population of pre-synaptic neurons to a mean membrane potential of a post-synaptic population, which in turn determines that population's output firing rate. A graphical description of a neural mass model can be seen in Figure 1.

To define a standard neural mass model, we begin by defining the post-synaptic potential of population n, $v_n(t)$, as a result of an input firing rate from population m, $g(v_m(t))$, as the convolution

$$v_n(t) = \frac{\alpha_{mn}}{\tau_{mn}} \int_{-\infty}^{t} h_{mn}(t - t') g(v_m(t')) \, \mathrm{d}t', \tag{1}$$

where α_{mn} is the gain for the post-synaptic response kernel (PSRK), denoted by $h_{mn}(t)$, from neural population m to n, and τ_{mn} is the membrane time constant. A common form of the PSRK is

$$h_{mn}(t) = \eta(t) t \exp\left(-\frac{t}{\tau_{mn}}\right), \tag{2}$$

where $\eta(t)$ is the Heaviside step function. An example of a PSRK can be seen in the left inset of Figure 1.

The firing rate of each population is related to the mean membrane potential by a sigmoidal activation function; the logistic function sigmoid is typically used:

$$g\left(v_n(t)\right) = \frac{1}{1+\exp\left(\varsigma_n\left(v_{0n}-v_n(t)\right)\right)}. \tag{3}$$

The quantity ς_n describes the slope of the sigmoid (approximating the variance of firing thresholds within the populations) and v_{0n} describes the mean firing threshold. An example of the sigmoidal activation function can be seen in the right inset of Figure 1.

The convolution in Equation 1 can also be written as the ordinary differential equation (ODE)

$$\mathrm{D}v_n(t) = \frac{\mathrm{d}^2 v_n(t)}{\mathrm{d}t^2} + \frac{2}{\tau_{mn}}\frac{\mathrm{d}v_n(t)}{\mathrm{d}t} + \frac{1}{\tau_{mn}^2}v_n(t) = \frac{\alpha_{mn}}{\tau_{mn}}g(v_m(t)), \tag{4}$$

where D is a linear differential operator. Equation 4 can be written as two coupled first-order ODEs by

$$\frac{\mathrm{d}v_n(t)}{\mathrm{d}t} = z_n(t), \quad \frac{\mathrm{d}z_n(t)}{\mathrm{d}t} = \frac{\alpha_{mn}}{\tau_{mn}}g(v_m(t)) - \frac{2}{\tau_{mn}}z_n(t) - \frac{1}{\tau_{mn}^2}v_n(t), \tag{5}$$

where $z_n(t)$ is a dummy variable. This forms the basis of a state-space model in a canonical format.

The parameters of the model, τ, α, v_0 and ς, can be set so the neural mass has characteristics of specific neural populations, such as pyramidal neurons, spiny stellate cells and fast and slow inhibitory interneurons ($\mathrm{GABA_a}$ and $\mathrm{GABA_b}$). The neural populations can then be connected to represent the circuitry of a cortical column and further to form networks of columns. Various kinds of neural mass models have been developed [1, 2, 3, 4], which are depicted in Figure 2. Each synaptic connection in these networks can be described by the 2^{nd}-order system of Equation 5, where the resultant dimensions of the networks of neural mass models in Figures 2 a, b and c are 6, 12 and 18, respectively. The parameters of the neural masses not only define the population type, but also the behaviour exhibited by the model. For example, certain parameter combinations result in a model of a cortical column that will generate alpha wave type activity (normal activity) and, for another set of parameters, we create a model that will exhibit epileptic behaviour [3]. Therefore, we consider a family of models, which we define generally as

$$\dot{\mathbf{x}}(t) = f_\theta\left(\mathbf{x}(t), \mathbf{u}(t)\right) \tag{6}$$

$$y(t) = \mathbf{C}\mathbf{x}(t) + e(t), \tag{7}$$

where $\mathbf{x}(t) \in \mathbb{R}^{n_x}$ is a state vector representing the postsynaptic membrane potentials generated by each population synapse and their time derivatives,

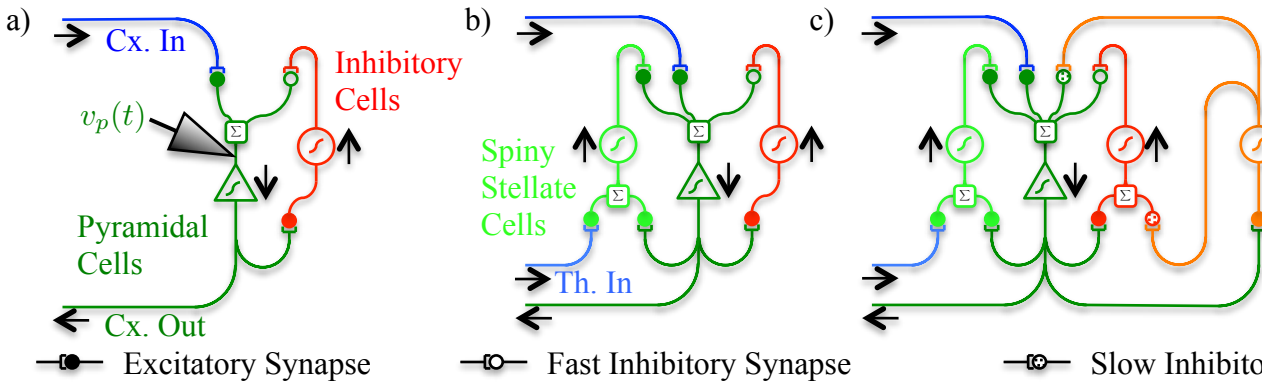

Figure 2: ***Models of Cortical Columns.*** *Each model has a pyramidal neural population, with various forms of feedback and inputs (Cx. = Cortex, Th. = Thalamus). The EEG is taken as the mean membrane potential of the pyramidal population, $v_p(t)$, with additive measurement noise. a) Minimal model of a column with inhibitory feedback [1]. b) An extension with excitatory feedback and afferent input from thalamus [2, 4]. c) Column with inhibition occurring across two times scales, enabling modelling of higher frequency activity observed in seizures [3].*

n_x is the number of states and $\mathbf{u}(t)$ represents the system input, which may be from afferent connections or other brain regions, exogenous input, or account for model inaccuracies. The function $f_\theta(\cdot)$ describes the dynamics, where $\theta \in \mathbb{R}^{n_\theta}$ determines the model type and the behaviour it exhibits. The iEEG is denoted by $y(t)$, $\mathbf{C}$ is the observation matrix, and $e(t)$ is the observation noise.

The model focused on in the following estimation sections is the formulation by Jansen and Rit [**?**]. Given space limitations we refer the reader to the article by Jansen and Rit[**?**] where the original state-space equations are presented. The parameters that were described in this section are related to the Jansen and Rit paper by

$$A = \frac{\alpha_{pe}}{2e_0c_1} = \frac{\alpha_{pi}}{2e_0c_3} = \frac{\alpha_{ep}}{2e_0c_2} = \frac{\alpha_{xp}}{2e_0} \tag{8}$$

$$B = \frac{\alpha_{ip}}{2e_0c_4} \tag{9}$$

$$a = \frac{1}{\tau_{pe}} = \frac{1}{\tau_{pi}} = \frac{1}{\tau_{ep}} = \frac{1}{\tau_{xp}} \tag{10}$$

$$b = \frac{1}{\tau_{ip}}, \tag{11}$$

where e_0 is a parameter that scales the maximum firing rate, A and B are synaptic gains for excitation and inhibition respectively, and a and b are the reciprocals of the synaptic time constants for excitation and inhibition, respectively, and the subscripts p, e, x, and i denote pyramidal (p), excitatory in-

terneuron (spiny stellate) (e), external (x) or inhibitory interneuron (i) populations, respectively. By making the assumption that all excitatory synapses share the same time constants and by defining the connectivity constants, c_1, c_2, c_3 and c_4, the network of neural masses is the JR NMM.

Present 3 models?

3 Theory / Performance Bounds

Introduce recursive computation of bound and Monte Carlo approximation

4 Methods?

5 Results

5.1 Effect of increasing model complexity – bound should increase

e.g. Bound for model A > model B > model C

5.2 Effect of parameters changing – bound should change

e.g. seizure easier to estimate than alpha rhythms

5.3 Effect of measurement noise? – pretty boring

e.g. scalp EEG vs iEEG?

5.4 Augmented models (joint estimation of parameters and states)?

6 Discussion

extensions to other applications e.g. experimental design

contextualize work w.r.t. existing literature?

7 Conclusion

References

[1] F. L. da Silva, A. Hoek, H. Smith, and L. Zetterberg, "Model of brain rhythmic activity," *Cybernetic*, vol. 15, pp. 27–37, 1974.

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[4] O. David and K. Friston, "A neural mass model for MEG/EEG: coupling and neuronal dynamics," *NeuroImage*, vol. 20, no. 3, pp. 1743–1755, 2003.